physicist. Without their aid the age of electricity would not have been possible, nor would we have the diagnostic and therapeutic uses of roentgen-rays or radium. The organic chemist has not only shed light on the complex processes occurring in the body, but has also produced many therapeutic agents. It is difficult for us to conceive of practicing without cocain, novocain, salicylic acid, acetanilid, eserin, pituitrin, salvarsan, and innumerable other drugs, all of which are of comparatively recent origin. A new era in the diagnosis and treatment of deficiency diseases was ushered in through the discovery, mainly by biochemists, of the vitamins which are so essential to life.

CIVILIZATION'S OBLIGATIONS TO RESEARCH WORKERS

This presentation has necessarily been brief, and many important findings have been omitted; likewise, much of the routine work has been overshadowed by the spectacular. However, we also wish to pay tribute to those individuals whose contributions have been of inestimable value to the medical profession. Some of the greatest contributions have been made by those who work single-handed while engaged in an active practice, and too often at great physical expense. In the present rather revolutionary period, many changes in our methods of practice have been suggested. Too often in time of turmoil the distractions outweigh the incentive, and it is impossible to progress with maximal efficiency. Research has been defined as "a method of keeping everyone reasonably dissatisfied with what he has." There never was a greater need for original investigation, or a better opportunity to salvage time for it than the present.

PURE SCIENCE INVESTIGATION AND CLINICAL OBSERVATION ARE BOTH IMPORTANT

Much has been written concerning the methods that should be employed in conducting original investigation, but it should be pointed out that, although there are some fields of study best left in the hands of the so-called pure scientists, research is not confined to the laboratory worker alone. Accurate observation of signs and symptoms, and their correlation with disease, will always be essential. In addition, there are certain phases of research which cannot be conducted by the laboratory investigator. The study of the earliest changes in disturbed function, which later develop into a definite clinical entity, lies solely within the realm of the family physician. Moreover, the science of prognosis and the evaluation of therapeutic remedies when applied to man necessitate an acuteness of observation during the progress of the disease which the scientists alone cannot appreciate. There can be no doubt that, although closely related, the fields to be conquered by laboratory methods and curative medicine are different. In order to progress with maximal efficiency, not only must observatory and experimental methods be coördinated, but there must also be an intimate contact between the investigators and the disease as it exists in man.

IN CONCLUSION

In conclusion, it seems to be in order to determine the results of all this knowledge which we have struggled so long and so diligently to obtain. One would be making a conservative estimate in stating that there are fifty million people in this country today whose lives have been saved or prolonged through medical science. While there are those who might question such an assertion, the figures are easily arrived at. One hundred years ago the average span of life was thirty-five years; today it is sixty. Since there are 126,000,000 people living in these United States, it is a simple problem to calculate the number of individuals whose existence at the present time is dependent upon the measures which have been instituted to prolong life. Moreover, seven years have been added to the average life span in the last twenty-three years, for in 1911 the average length of life was only fifty-three years. This also means that there are 13,000,000 people alive today who would have been dead but for the progress made in medical science. At the present rate of increase in knowledge, man can look forward to an average of sixty-five years of life by 1944. The battle in which we are active participants will never cease, for the diverse ramifications of our science are bound to hold our interest. However, it is the judicious employment of these days that will continue to advance the front of our knowledge during this period, and speed it on its way to even greater heights.

The Mayo Foundation.

THE DIFFERENTIAL DIAGNOSIS OF INTRACRANIAL DAMAGE*

By E. J. MORRISSEY, M. D.
San Francisco

DISCUSSION *by Howard C. Naffziger, M. D., San Francisco; Carl W. Rand, M. D., Los Angeles; Howard W. Fleming, San Francisco.*

THE symptoms and signs of cerebral injury may result not only from intracerebral lesions, namely, concussion, contusion, laceration and intracerebral hemorrhage, but also from cerebral compression due either to depressed fractures, extradural or subdural hemorrhages.

DIFFERENTIAL DIAGNOSIS

The differentiation between these conditions is the essential point in determining the method of treatment.

Concussion.—Unfortunately in the past the term concussion has been used in a very elastic manner to include severe cerebral contusion and even laceration.

At present it is customary to designate as concussion head injuries in which no definite pathological changes are apparent.

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* From the surgical service of Stanford University, San Francisco County Hospital, Department of Public Health.

Read before the General Surgery Section of the California Medical Association at the sixty-third annual session, Riverside, April 30 to May 3, 1934.

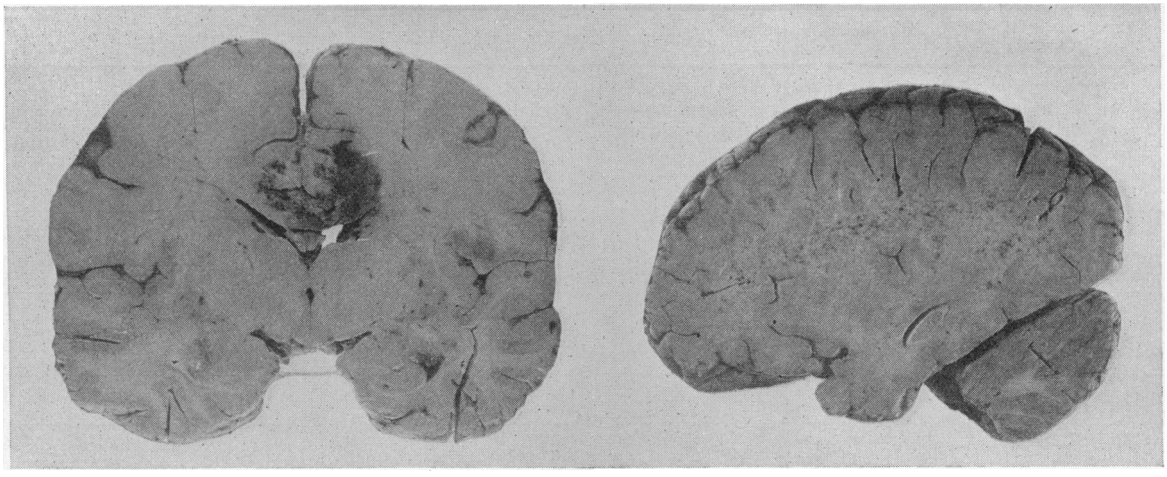

Fig. 1.

Fig. 2.

The term, however, will probably be discarded. I am certain that as our methods of investigation advance we will be able to demonstrate pathological changes in those specimens that are now considered as normal.

As a result of the studies of Cajal and others, this has already in part been accomplished.

Contusion.—Contusions of the brain may vary from a few petechial hemorrhages to extensive bruising of the cortex and underlying tissues.

The petechial hemorrhages have a tendency to group themselves about localized areas. (See Fig. 1.) This grouping is one of the distinguishing points between petechial hemorrhages resulting from trauma and those following emboli, infection, etc. (See Fig. 2.)

Laceration.—Laceration of the brain is a definite tearing of the tissues, and is naturally associated with hemorrhages into the surrounding brain tissue, and usually into the subarachnoid and subdural spaces.

Lacerations may be found at any point, but they are especially common over the bases of the frontal and temporal lobes. They may be directly under the point of injury, but are usually *contrecoup.*

Hemorrhage.—Extracerebral hemorrhage may be either extradural, subdural (acute or chronic), or subarachnoid.

Extradural Hemorrhage. — Extradural hemorrhage practically always results from a rupture of the main trunk or one of the branches of the middle meningeal artery.

In rare instances it occurs from either a tearing of one of the large venous sinuses or a separation of the dura from the skull at the time of the accident, with bleeding from the bone.

The bleeding from the torn vessel or vessels strips the dura from the skull, and as the hemorrhage enlarges symptoms of compression ensue.

Extradural hemorrhages, as a rule, are disc-shaped, thin at the margins and thick in the center. They may be of any size, varying in weight from a few to over two hundred grams.

Moody, in one hundred autopsies, found the average weight of the clot in fatal cases to be one hundred and ten grams.

The majority of extradural hemorrhages occur in patients between the ages of twenty and fifty. They are rare in childhood and elderly people. In Vance's series of sixty-one cases only three occurred before the age of twenty.

As a general rule the symptoms resulting from these hemorrhages make their appearance within the first twenty-four hours. The time interval may vary, however, from a few hours to several days, depending entirely on the rapidity with which the dura is stripped from the bone.

Subdural Hemorrhages.—When the force of the blow is sufficient to cause either severe cerebral contusion or laceration of the cerebral veins with tearing of the arachnoid, we have bleeding into the subdural space.

These subdural hemorrhages may be either unilateral or bilateral, and likewise may vary in size from a small localized clot to a large hemorrhage sufficient to cause marked cerebral compression.

Vance, in 512 autopsies, felt that death was due to cerebral compression by a large collection of dural blood in 132 cases. The average weight of the clot was 61.1 grams.

Chronic Subdural Hematomas.—Chronic subdural hematomas differ from the acute subdural hemorrhages spoken of above and lie, as Virchow was the first to show, in the subdural space. They are surrounded by a definite capsule and are often bilateral.

Trotter and others believe that the bleeding is due to injury of the veins passing from the brain to the tributaries of the superior longitudinal sinus.

In the great majority of cases they occur some time after what was apparently a very minor head trauma. This period, between the injury and the development of symptoms, may vary from a few weeks to several months or even years. Often the initial injury is so slight and the elapsed time before the start of symptoms so long that it is entirely forgotten.

TABLE 1.—*Differential Diagnostic Points*

	Contusion Laceration	Extradural Hemorrhage	Chronic Subdural Hematoma
Unconsciousness	Immediate	Stupor coming on after a latent period. If initial injury severe latent period may be absent.	Latent period varies from few weeks to many months.
Temperature	Fall at first due to shock. Rise following depending on severity. When above 104 degrees recovery rare.	No rise.	No rise.
Pulse and Respirations	Rapid in shock. Slow in medullary involvement. Rapid in medullary failure.	Slow.	Slow.
Blood Pressure or Pulse Pressure	Increase with medullary involvement.	Increased.	No increase.
Spinal Fluid	Contains blood—amount depends on severity. Pressure varies.	No blood unless contusion or laceration present. Pressure increased.	Either slightly blood tinged, xanthochromic or clear.
Neurological Findings			
(a) Weakness or paralysis	Immediate.	Late and progressive.	Gradual and progressive. Occasionally on same side as hemorrhage.
(b) Pupils	Very dilated on side of lesion. Dilated and fixed prognosis poor.	Dilated as a rule on side of hemorrhage.	Often dilated on side of hemorrhage.
(c) Ophthalmoscopic	Negative first 24 hours at least.	Negative.	Bilateral choked discs usually present; greater on side of hemorrhage.
X-ray Findings	Fracture may or may not be present.	Fracture line crossing middle meningeal groove usually present.	Usually no fracture. Pineal shifted.

Following the latent period we have the history and findings of gradually increasing intracranial pressure, namely, headache, nausea and vomiting, visual disturbances, choked discs, etc. In view of these facts, it is extremely important in any case of gradually increasing intracranial pressure to go carefully into the past history.

Subarachnoid Hemorrhages.—Bleeding into the subarachnoid spaces occurs in practically all cerebral contusions of any severity.

It varies from merely a slight pinkish discoloration of the cerebro-spinal fluid to the point where it is practically pure blood.

Essick, Bagley, Fay and others believe that this condition accounts for adhesions and blockage of the circulation of the cerebro-spinal fluid, and many of the post-traumatic cerebral symptoms.

Subdural Collections of Cerebro-Spinal Fluid.—A discussion of the differential diagnosis of intracranial lesions would not be complete without mention of the localized subdural collection of cerebro-spinal fluid first spoken of by Naffziger. These cases present symptoms and findings very similar to extradural hemorrhages.

SPECIAL SYMPTOMS

Unconsciousness. — The duration of the period of unconsciousness immediately following a head injury is in direct proportion to the severity of the cerebral damage.

The general rule may be laid down that the longer the period of unconsciousness, the more severe the cerebral contusion or laceration and the more guarded must be the prognosis.

Occasionally we see deaths resulting in patients who were not unconscious following the accident. In these cases it is practically always due to some complication, such as subdural or extradural hemorrhage, meningitis, etc. There is, of course, the occasional case of continued bleeding into the cerebral tissues following a laceration.

Stupor, coming on gradually after a period of consciousness, is practically always the result of an extradural hemorrhage. The period of consciousness is spoken of as the latent period and may vary from a few minutes to hours or even days.

It must always be borne in mind, however, that the initial period of unconsciousness due to the contusion may be of sufficient length that it overlaps the unconsciousness resulting from the hemorrhage, and that, therefore, there will be no latent period.

Temperature.—The temperature rise within the first twenty-four hours is likewise a fair indication of the amount of cerebral contusion or laceration. In the average case, immediately following the injury, there is a fall in temperature, the result of shock followed by a rise above normal.

In those cases in which recovery takes place the average varies between 101 and 103 degrees. As a rule recovery seldom occurs when, as a direct result of the cerebral trauma, the rise is above 104 degrees.

Pulse and Respirations.—The point has always been emphasized that slowing of the pulse and respirations, especially when associated with rise

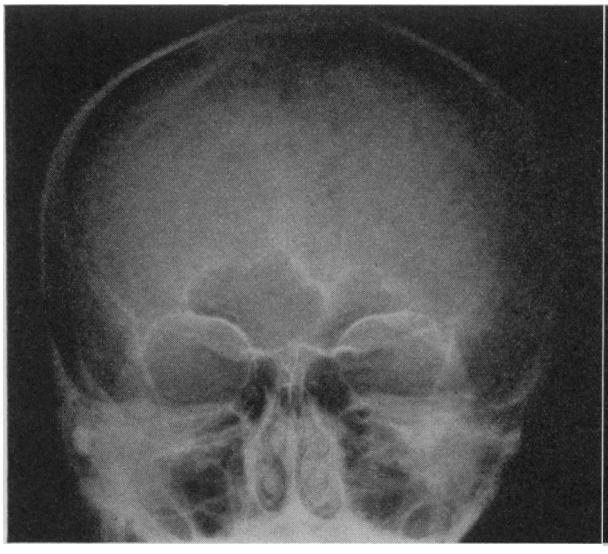

Fig. 3.

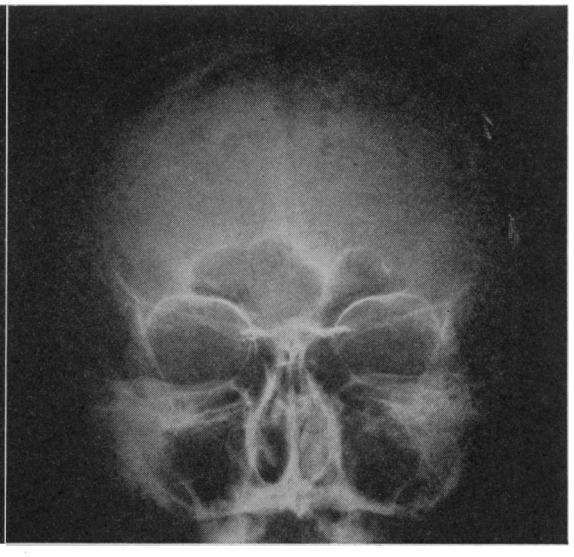

Fig. 4.

in blood pressure or a widening pulse pressure, is indicative of increased intracranial pressure.

This is not the entire truth. What this syndrome indicates is medullary involvement.

Before this was realized, one occasionally was surprised, at operation or postmortem examination, to find the brain shrunken and fallen away from the dura in those cases that had marked slowing of pulse and respirations. The measurement of the spinal fluid pressure is a good indication as to whether or not these medullary symptoms are due to increased intracranial pressure.

It is extremely important to bear the above point in mind when the question of dehydration is considered in the treatment of head injuries.

Slowing of pulse and respirations, with a rise in blood pressure or widening pulse pressure, when due to increased intracranial pressure is a result of either brain edema or hemorrhage.

If it occurs within the first twenty-four hours it is most likely due to an extradural hemorrhage. In Vance's series of postmortem examinations, on 106 cases of extradural hemorrhage, death occurred in over 50 per cent within the first twenty-four hours.

When the symptoms make their appearance after the first twenty-four hours they are more likely to be the result of cerebral edema, secondary to contusion or laceration.

On the other hand, when we have these findings occurring weeks or months after an injury, we may with certainty make a diagnosis of a subdural hematoma.

NEUROLOGICAL EXAMINATION

A careful neurological examination made immediately following the accident is the best method of distinguishing between lesions due to contusion or laceration and those resulting from hemorrhage.

If immediately following cerebral trauma there is a weakness or paralysis with definite reflex changes, then it is no doubt the result of contusion or laceration.

On the other hand if the first examination is negative and subsequent findings reveal a gradually developing paresis, especially starting about the face, then it is most probable that we are dealing with an extra- or intradural hemorrhage. Occasionally, however, this picture is presented by increasing edema about a laceration or an enlarging subcortical hemorrhage.

The pupillary findings are extremely important. They are valuable both from a prognostic and diagnostic point.

Patients with bilaterally dilated and fixed pupils very seldom recover. In Blakslee's series of 610 cases of fracture of the skull with bilateral dilated and fixed pupils, 95.5 per cent died. The existence of previously fixed pupils must, of course, be kept in mind.

Unilateral dilatation with fixation of the pupil occurs as a rule on the same side as the cerebral involvement and may be due to either cerebral contusion or extradural hemorrhage.

A gradually developing unilateral dilatation of the pupil is more likely the result of an extradural hemorrhage. Occasionally it is the only positive localizing sign. This point has been emphasized especially by Holman and Scott, Lyerly and others.

Evidence of choking of the optic discs is extremely rare in the first forty-eight hours, even though there be fairly marked increased intracranial pressure. Ophthalmoscopic studies should be made as soon as possible, because then we are in a position to note the early signs suggesting increased intracranial pressure such as engorgement and tortuosity of the veins, blurring of nasal margins and hyperemia of the discs. A choking of the discs, on the other hand, is a common finding in chronic subdural hematoma.

A mydriatic should not be used within the first few days because one is then unable to follow pupillary changes which are extremely important.

SPINAL PUNCTURE

Measurement of the pressure and the examination of the spinal fluid is extremely important. Blood in the spinal fluid is found in practically every cerebral contusion of any severity and in all lacerations. As a rule the more severe the cerebral injury the higher the percentage of blood in the spinal fluid. Likewise the more guarded must be the prognosis.

If clear spinal fluid is obtained in a patient presenting the signs and symptoms of increased intracranial pressure then we are more than likely dealing with an extradural hemorrhage.

It does not follow, however, that the presence of blood in the spinal fluid rules out an extradural hemorrhage. Very often cerebral contusion is also present.

The diagnosis of meningitis is likewise verified by examination of the spinal fluid. It must be remembered, on the other hand, that blood in the spinal fluid, especially in the region of the posterior fossa, will cause the usual signs suggesting meningitis, namely, stiffness of the neck and bilateral Kernigs.

ROENTGEN-RAY FINDINGS

Everyone recognizes that there is no direct relationship between the degree of brain injury and the presence or absence of a fracture, and that patients who are in extremely poor condition or in shock should not be subject to roentgen studies. However, we often obtain valuable information which enables us to make a diagnosis.

Extradural hemorrhages occur in the absence of fracture, yet they are much more likely to be present with a fracture crossing the grooves of the middle meningeal artery or its branches. Occasionally, in an unconscious patient, the presence of a fracture crossing one of these grooves is the most important deciding and localizing factor.

What has been a great aid to us in making a diagnosis of a hemorrhage either extradural or subdural is the shifting of the pineal body. It has been useful especially in those cases in which the hemorrhage is on the same side as the symptoms. Fig. 3 illustrates the shifting of the pineal in a large subdural hemorrhage and Fig. 4 the return to normal position after removal of the hemorrhage.

Occasionally we find, associated with cerebral contusion or laceration, a hemorrhage either extra or intradural and of sufficient size to in itself produce symptoms of cerebral compression. These cases are extremely difficult to diagnose, because the symptoms and findings of one tend to mask those of the other.

Le Count and Apfelbach, in reporting a series of extradural hemorrhages, made the statement that only 52.26 per cent were of sufficient size to cause death; and as stated, Vance, in 512 autopsies, felt that death was possibly due to cerebral compression by a collection of subdural blood in 132 cases.

When the above is encountered the question always arises as to whether or not a certain percentage of those cases would have recovered from the cerebral damage were the extra burden of the hemorrhage removed.

A diagnosis, if it is to be made, demands careful observation with frequent neurological examination for evidence of change. One has no right to assume, because he finds bloody spinal fluid and other evidence of cerebral contusion or laceration, that all the ensuing symptoms are a result of this lesion.

If there is any question, trephine openings should be made. By our present methods there is no shock associated with this procedure, and if a hemorrhage is found and evacuated, the patient has a far better chance to recover.

I would rather operate on a patient and not encounter a hemorrhage than to find at postmortem that a hemorrhage had been overlooked.

909 Hyde Street.

DISCUSSION

HOWARD C. NAFFZIGER, M.D. (University of California Medical School, San Francisco).—The statistical points quoted by Doctor Morrissey are helpful in forming our clinical judgment and in prognosis. The management of craniocerebral injuries does not require particularly refined methods of neurological examination, nor does the state of the patient usually permit them.

Proper interpretation of the physiological disturbances of consciousness, of respiration, heart rate, and blood pressure are of paramount importance. By these and the gross neurological manifestations, our treatment is guided.

The physiological responses to acute cerebral compression have been well established in laboratory experimentation. In our patients, however, these well-known alterations may be lacking, and we may find bizarre responses to brain compression. For example, an increasingly rapid pulse may be associated with a rising intracranial pressure instead of the expected slow pulse. In such instances we are dealing with the reactions of a traumatized and contused brain—one which behaves differently from the untraumatized but otherwise normal brain, which is being subjected to gradual compression in the experimental animal.

Doctor Morrissey's presentation makes clear, I think, that only a small proportion of patients with craniocerebral injuries are benefited by operation.

❦

CARL W. RAND, M. D. (523 West Sixth Street, Los Angeles).—Doctor Morrissey has given a very excellent differential diagnosis of the various types of intracranial hemorrhages following head injury. His reference to the pineal shift in certain cases of chronic subdural hematoma and of large subdural hemorrhages is timely. It is felt that x-ray plates should be studied more carefully for the possibility of pineal shift in suspected cases of intracranial hemorrhage.

❦

HOWARD W. FLEMING, M. D. (384 Post Street, San Francisco).—Doctor Morrissey's paper on "The Differential Diagnosis of Intracranial Damage" is most timely.

No diagnostic problem is more open to controversy. Recently, well-known authorities have expressed their opinions as to the diagnosis and treatment of head injuries in a most dogmatic manner. Diametrically opposed methods have been advocated so forcefully that the medical profession is at a loss to know how to treat their cases. Only too frequently this indecision leads to procrastination, and opportunities for effective therapy are overlooked.

Judicial use of all recognized methods will result in a far greater percentage of correct diagnosis. There

is no substitute for a careful history followed by frequently repeated examination. Spinal puncture and x-rays often are helpful. Each case is an individual problem, but careful evaluation of all information to be obtained usually suggests the nature and extent of the intracranial pathology.

The methods and conclusion, as given by Doctor Morrissey, are those generally accepted by the great majority of neurological surgeons.

DOCTOR MORRISSEY (Closing).—In conclusion, I wish to thank the various men who have discussed this paper, and emphasize the fact that each case is individual in itself, and that one should not adhere too closely to any general rule.

URETHRAL STRICTURES—A RÉSUMÉ OF TREATMENT*

By WILBUR B. PARKER, M. D.
AND
CHESTER H. MACKAY, M. D.
Los Angeles

DISCUSSION *by Elmer Hess, M. D., Erie, Pennsylvania; Henry A. L. Kreutzmann, M. D., San Francisco; Albert M. Meads, M. D., Oakland.*

THE great amount of current literature on the diagnosis of urethral strictures leads us to believe that the procedures of our preceptors were, and are today, just as accurate, and in many instances more accurate than the teachings of more recent authors. The older methods comprehended at the start an accurate clinical history, palpation, and a soft rubber catheter.

PROCEDURE WITH OBSTRUCTIONS

When obstructions are met by these simple methods, the following procedures must be resorted to:

Calibration of the urethra by use of button-sounds and button-bougies. Further, in attempting to calibrate the urethra, a size number 6 or larger should be used first. If unable to pass any of these sizes, smaller types should be attempted, down to a filiform, to be used if necessary. By this method the size, number and location of strictures can be determined.

Gentle instrumentation should be the watchword for all urologists, in diagnostic or treatment procedures. The less traumatism, the less the possibility of occurrence of complications. This rule should apply to all classes of strictures, both in male and female patients, and irrespective of their locations.

Urethrograms, whatever the opaque medium used, have been made much of in medical literature, to assist in the diagnosis and location of urethral strictures, regardless of fallacious interpretations. We must admit that a great many of the newer researches have had a scientific playground. Urethral strictures are as plentiful in number at the present time, as they have been in the past, and will continue to be so long as the human race may exist.

*Read before the Urology Section of the California Medical Association at the sixty-third annual session, Riverside, April 30 to May 3, 1934.

DEVELOPMENT OF URETHRAL STRICTURES

Time elements in the development of urethral strictures may be classified as: (1) Congenital, designated by the term that now exists and a type that does not contract; (2) Traumatic; (3) Inflammatory.

Urologists should hope to see the day when no adolescent or adult, male or female patient with a congenital stricture, will apply for treatment for a urethritis, specific or nonspecific. Without doubt they are the most unfortunate class of victims, including urethral chancre, that we have to treat today.

Traumatic strictures depend upon the amount of damage done to the urethra at the time of accident. In comparison with strictures of inflammatory origin, time of formation is much shorter. Inflammatory strictures contract slowly, depending upon the severity of the urethral infection and whether there have been any complications of the urethritis. It has been said that about one-third of inflammatory strictures occur within one year after infection, while two-thirds or more occur within two to four years.

The most frequent sites of congenital strictures are at the external meatus. Inflammatory strictures occur mostly in the phallic, bulbous and membraneous urethras, rarely in the posterior urethra. Spasmodic strictures have been denied as to whether they ever occur in the phallic and membraneous urethras. Stricture plus spasm lead to acute retention, an entity which we cannot deny. Traumatic strictures occur any place along the site of trauma, and this rule holds true in female strictures, despite the anatomical differences between the two sexes.

TREATMENT

Surgical and non-surgical treatment in private and institutional practice is usually somewhat different.

Patients in private practice are instructed to report as often as needed, in order to carry them from a filiform size to a No. 22 or No. 24 French-size dilator. Progress is often slow, and very often in the contracting types a period of six months or over is required to accomplish a successful result. Patients with filiform types of strictures are advised to report daily, until such times that it is possible to use other than filiforms. Following the filiform treatments for gradual slow dilatation, small bougies are used. In our experience, this method has caused less disturbance to the voiding ability of the patient.

Institutional patients are generally those that have been badly traumatized, and are suffering from acute retention. Such patients should be given indicated sedatation, for they rarely tolerate satisfactorily a local anesthesia, owing principally to spasm or complete occlusion at points of obstruction. For such patients, attempts are made to pass some type of catheter, preferably a Coude or natural curve type. Hospitalization usually follows, pending the reaction of the patient to relief and dilatation. If response to treatment is favorable—meaning reasonable ability to void—